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Myocardial infarction

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Contents

Objectives

- If a person at risk of a myocardial infarction has an acute coronary syndrome lasting over 20 minutes, an imminent myocardial infarction must be suspected. Instead of chest pain, acute dyspnoea may be the primary symptom.
- An acute coronary syndrome without myocardial damage is often unstable angina, which calls for active treatment.
- The diagnosis should be made without delay since early therapy improves the prognosis decisively.
- Thrombolytic therapy is given as early as possible in all cases with a clinical picture of imminent myocardial infarction and corresponding ECG changes (See related EBM Guideline: **Thrombolytic therapy in acute myocardial infarction** available on the EBM Web site).
- Acute angioplasty (PTCA, PCI) is an alternative or a complementary procedure to thrombolytic therapy (Level of Evidence= A; Evidence Summary available on the EBM Web site).
- Health care system should include a planned care pathway for coronary patients.

Diagnosis

- The diagnostic criteria change in the course of treatment.
    - During first aid, pain is the primary symptom.
    - When thrombolytic therapy is considered, an ST change on the ECG or a recent left bundle branch block (LBBB) should be taken into account (See related EBM Guideline: **Thrombolytic therapy in acute myocardial infarction** available on the EBM Web site).
    - In addition to pain and ECG findings, myocardial enzyme levels are needed for definite clinical diagnosis.
    - Various registers and studies need all the information mentioned above, plus autopsy findings. In these cases, the terms "definite" and "possible" infarction are used.
- For differential diagnosis of chest pain see related EBM Guideline: **Differential diagnosis of chest pain** available on the EBM Web site.
- The pain in myocardial infarction lasts over 20 minutes and is localized widely in the retrosternal area, with radiation to the arms, back, neck or lower jaw. The pain is squeezing, is experienced as tightness, heaviness and pressure or pressing. Breathing or changing posture does not influence the intensity of pain. The pain is usually severe and consistent. It may be localized in the upper abdomen, in which case, if nausea and vomiting are also present, it simulates acute abdominal disease. The patient is often pale, in a cold sweat and serious.
- Myocardial infarction may also occur as acute pulmonary oedema, spells of unconsciousness or sudden death.
- Thrombolytic therapy is indicated
    - if the pain has lasted less than 6 - 12 (24) hours and there is at least a 2-mm elevation in the ST segment in at least two chest leads, or
    - a 1-mm elevation of ST in at least two leads in the extremities, or
    - a reciprocal ST depression in V1 - V4, or
    - a recent LBBB.
- The contraindications for thrombolytic therapy must always be considered (See related EBM Guideline: **Thrombolytic therapy in acute myocardial infarction** available on the EBM Web site).
- In clinical investigation, remember that the ECG and myocardial markers change with the course of the disease: first there is an ST elevation, after that development of the Q wave, and finally T-wave inversion. Complications must also be recognized. In a T-wave infarction (non-Q-wave infarction), no classical Q waves are present, but the diagnosis is based on an increase of myocardial enzymes, chest pain, or ST-T changes. Classical Q-wave changes, ST elevations and T inversions may be caused by various other diseases, which should be remembered in the differential diagnosis. An old infarction, bundle branch block and early repolarization make the diagnosis difficult, in which case the change in ECG is important and an old ECG recording valuable. When added to other criteria, "minor" signs of infarction are also important.
- The European Society of Cardiology and the American College of Cardiology have agreed on a new definition of myocardial infarction[1]:
    - Typical increase in the concentration of serum cardiac troponins or CK-MB associated with at least one of the following:
        - symptoms of cardiac ischaemia
        - recent pathological Q waves in the ECG
        - ischaemic ST segment changes in the ECG
        - coronary artery revascularization.

ECG diagnosis

- Points for taking an ECG: acute care, emergency room, 12 hours later, on day 2, upon discharge from hospital and thereafter as deemed necessary.
- ECG is the most important diagnostic procedure. To start with, the positions of the chest leads must be marked on the skin to allow detection of meaningful changes on the ECG. By monitoring the ECG, the efficacy of the treatment can be assessed. However, in the early stages there may be no changes in ECG, and the changes may be first evident after hours or even days. An ECG diagnosis is made more difficult by an old infarction, LBBB or posterior infarction.
- In posterior wall infarction, a reciprocal ST segment depression in V1 - V4 simulates ischaemia. A posterior infarction is, however, often inferoposterior and, in addition to ST segment depression, ST segment elevations are found in leads III and aVF.
- ST depression is suggestive of ischaemia and/or unstable angina pectoris. Extensive ST depressions in connection with a clinical picture of myocardial infarction can indicate subendocardial damage.

Tests following the ECG

- Troponin is the most important new marker and is replacing CK. - CK and CK-MB or CK-MB mass
- A negative troponin T, troponin I or CK-MBm result 9 - 12 hours after the onset of symptoms practically rules out myocardial infarction.
- Troponin T test is also valuable, if the time lapse since the beginning of the symptoms is more than 24 hours (the concentration remains elevated longer than that of CK). An elevated troponin T or troponin I concentration predicts adverse events irrespective of ECG findings (Level of Evidence=A; Evidence Summary available on the EBM Web site).
- The tests should be performed 3 times in case of suspected infarction: on arrival of the patient and 12 and 24 hours after arrival.
- Blood haemoglobin, leukocytes, ESR and CRP
- Serum sodium and potassium, and chest x-ray if needed

Troponin-T or troponin-I

- Principal indicators of myocardial damage, which can also be determined by means of rapid testing methods suited for primary health care. A reading device facilitates the interpretation.
- Troponin is more myocardium-specific than CK-MB and is also very sensitive.
- The concentration increases rapidly (in 4 - 6 hours) after myocardial damage, and the elevated levels persist for at least one week.
- Indications:
    - To verify or exclude myocardial infarction (or myocarditis) when at least 6 hours have elapsed from the onset of pain. Unstable angina pectoris may give positive results, indicating slight myocardial damage, which means that the prognosis is serious regardless of the ECG findings and active treatment is necessary. The normal reference concentration is zero.
    - A negative result within 12 hours after the onset of pain excludes infarction.
    - Also used for the diagnosis of infarction when the patient´s arrival for treatment is delayed, and CK and AST have returned to normal.
    - Troponin verifies myocardial infarction in cases where high CK concentration from skeletal muscle increases the CK-MB concentration over normal limits.

Serum CK-MB mass

- More specific and sensitive than CK-MB.
- Abnormal within 6 - 8 hours from the beginning of the pain, and remains abnormal for 1 - 2 days.
- Slightly positive values may indicate mild myocardial damage that requires active treatment. Unlike with troponin, the normal concentration of CK-MB is not zero. There is an uncertain borderline area of 5 - 10 µg/L between the positive and negative result.

Myoglobin

- Reacts most rapidly to myocardial damage and is positive from the first hours onward.
- Not a specific indicator of myocardial damage. Negative myoglobin is valuable in exclusion diagnosis.
- Lack of reference values limits use.

Differential diagnosis

- The most important differential diagnoses include
    - myopericarditis (See related EBM Guideline: **Myocarditis** available on the EBM Web site)
    - aortic dissection (See related EBM Guideline: **Aortic aneurysms and dissection** available on the EBM Web site)
    - pulmonary embolism (See related EBM Guideline: **Pulmonary embolism (PE)** available on the EBM Web site)
    - unstable angina pectoris (See related EBM Guideline: **Unstable angina pectoris** available on the EBM Web site)
    - oesophageal pain (See related EBM Guideline: **Heartburn** available on the EBM Web site).
- See also article on differential diagnosis of chest pain (See related EBM Guideline: **Differential diagnosis of chest pain** available on the EBM Web site).

Treatment

- Oxygen, if there are problems in oxygenation (pulmonary oedema).
- For treating pain
    - Glyceryl nitrate: mouth spray or sublingual tablet
    - Morphine 4 - 6 mg i.v., additionally 4 mg 1 - 3 times at 5 min intervals, if necessary. Oxycodone 3 - 5 mg i.v. is an alternative.
    - A beta-blocker (metoprolol, atenolol, practolol) 2 - 5 mg i.v. may sometimes ease the pain.
- ASA 250 mg, chewable tablet or dissolved in water, unless there are contraindications (active ulcer, hypersensitivity to ASA, anticoagulation) (Level of Evidence=A; Evidence Summary available on the EBM Web site).
- A beta-blocker is always instituted, unless there are contraindications (asthma, hypotension, heart insufficiency, conduction disturbance, bradycardia). The first dose can be given intravenously if the patient is in pain, or orally if the patient is pain-free and time has passed

since the infarction. Beta-blockers are useful especially in patients who are tachycardic and hypertensive but do not have heart failure.
    - i.v. dose: metoprolol or atenolol 5 mg.
    - Orally, metoprolol or atenolol 25 - 50 mg x 2
- Thrombolytic therapy, unless there are contraindications (See related EBM Guideline: **Thrombolytic therapy in acute myocardial infarction** available on the EBM Web site) (Level of Evidence=A; Evidence Summary available on the EBM Web site).
- Immediate PTCA (Level of Evidence=A; Evidence Summary available on the EBM Web site) may be performed when thrombolytic therapy is contraindicated. May be more effective than thrombolysis.
- An ACE inhibitor in the acute phase of infarction (< 24 hours) to all patients with signs or symptoms of heart failure, anterior wall infarction or reinfarction (Level of Evidence=A; Evidence Summary available on the EBM Web site).
    - E.g. captopril. Start with 6.25 mg and increase the dose rapidly.
- Continuous nitrate therapy (Level of Evidence=A; Evidence Summary available on the EBM Web site)
    - Administered as an infusion, if the patient has ischaemic pain and pain medication has no effect. Nitrate infusion (See related EBM Guideline: **Nitrate infusion in angina pectoris and myocardial infarction** available on the EBM Web site).
    - Orally, e.g. isosorbide dinitrate 10 - 20 mg x 2 - 3.
- Heparinization is often indicated, if the patient
    - needs prolonged bed rest and is clearly obese (thrombosis prophylaxis)
    - has atrial fibrillation (also permanent warfarin therapy)
    - has ventricular aneurysm (also permanent warfarin therapy)
    - has unstable angina pectoris
    - has embolic complications

Arrhythmias in myocardial infarction

Objectives

- To prevent sudden death and treat severe arrhythmias immediately.
- To prevent arrhythmias by treating the underlying conditions.

Causes of arrhythmias

- Myocardial damage, ischaemia and sympathetic stimulation cause ventricular arrhythmias.
- Ejection failure causes supraventricular tachyarrhythmias and atrial fibrillation.
- Vagal stimulation causes bradyarrhythmias and AV conduction disturbances, especially in cases of inferior-posterior wall infarction.
- Reperfusion often causes benign ventricular rhythm but also severe ventricular arrhythmias.

Ventricular fibrillation

- Often occurs within 2 - 4 hours of infarction. After 12 hours, a primary ventricular fibrillation is rare.
- An early ectopic beat may initiate ventricular fibrillation in an ischaemic myocardium. Ectopic beats are not treated if cardiac monitoring is effective.

- Treatment
    - Acute ventricular fibrillation is treated by immediate defibrillation starting with 200 joules. Prolonged ventricular fibrillation frequently calls for cardiopulmonary resuscitation (CPR).
    - To prevent recurrence of fibrillation, lidocaine is given: initially as bolus of 100 mg, which can be repeated if necessary. Thereafter, a continuous infusion of 3 - 4 mg/min is given. Amiodarone is an effective alternative to lidocaine.
    - A beta-blocker is usually added to the therapy.

Ventricular tachycardia

- More than three ectopic beats and a heart rate over 120 beats/min.
- Brief, spontaneously ending spurts are seen in over 50% of patients with infarction during the first two days. They occur mainly after 8 - 14 hours, not immediately after the infarction, as ventricular fibrillation does.
- Ventricular tachycardia leads to haemodynamic collapse or ventricular fibrillation. The severity depends on the duration, variability, frequency and timing of tachycardia.
- Treatment
    - Beta-blocker
    - Lidocaine boluses and infusion as in ventricular fibrillation, if haemodynamics is compromised. Amiodarone is a good alternative.
    - If necessary, synchronized cardioversion shock with 50 joules is performed.
    - Late in infarction, ventricular tachycardia is, like ventricular fibrillation, a serious problem that requires further examination.

Ventricular ectopic beats

- Occur in nearly all patients with painful myocardial infarction.
- May cause complications if they are frequent (more than 5/min), are variable or occur concomitantly with an early T wave.
- Treatment is usually not necessary if cardiac monitoring is effective. A beta-blocker may be indicated.

Idioventricular rhythm

- Natural ventricular rhythm is an arrhythmia often associated with myocardial infarction. In the reperfusion phase, it may even indicate that thrombolysis has been successful. The frequency is often economical 70 - 80 bpm and drug therapy is not necessary.

Supraventricular tachyarrhythmias

- Atrial fibrillation in a patient with infarction is often associated with cardiac insufficiency and worsens the prognosis. Atrial fibrillation multiplies the risk of stroke, which is why LMW heparin and warfarin therapy are indicated.
- Atrial fibrillation is often associated with the thrombosis of the right coronary artery or the circumflex branch: reperfusion often corrects also atrial fibrillation.
- Atrial function is important in myocardial infarction. In cardiac insufficiency, rapid atrial fibrillation requires active direct current (DC) cardioversion. Often, the achieved sinus rhythm

does not last. In such a case, haemodynamics must be stabilised (oxygenation, treatment of pulmonary oedema, controlling of ventricular response with a beta-blocker and digitalis) after which spontaneous reversal of the rhythm is waited for. The effect of the beta-blocker is seen rapidly but that of digitalis not before several hours. Rapid ventricular response may be controlled even if cardiac insufficiency is present: the benefit often outweighs the disadvantage.
- Selective beta-blockers are best suited for maintaining the achieved sinus rhythm.
- Intravenous amiodarone will not reduce the contraction of the myocardium. It is effective in prophylaxis of atrial fibrillation (together with a beta-blocker) and it may be used in cardioversion of atrial fibrillation and/or slowing down the ventricular response.
- Ibutilide is a new class III drug with a single indication: treatment of atrial fibrillation and flutter. There are limited data on its use in patients with infarction.
- Note! A broad QRS complex tachycardia in a patient with infarction must always be treated as a ventricular tachycardia.

Bradyarrhythmias

- A strong vagal reaction in the early stages of infarction may lead to a circulatory collapse.
- Postero-inferior wall infarction is often associated with a functional AV block. The QRS complex is narrow and the heart rhythm is 50 - 60 even in cases of a total block. A pacemaker is rarely needed.
- In anterior wall infarction, the conduction system is blocked: the QRS complex is wide, the substituting rhythm is slow (30 - 40), the patient is in a poor condition and pacing is necessary.
- Drug treatment
    - atropine 0.5 mg i.v., repeated as necessary, for treatment of functional bradycardia.

Pacemaker

- In anterior wall infarction pacing is indicated if there is a II or III degree block. Pacing should be anticipated in case of a trifascicular block, alternating right and left bundle branch block, or if an extensive infarction is associated with LAHB or LPHB.
- Postero-inferior wall infarction associated with a III degree AV block requires pacing if bradycardia is detrimental to haemodynamics and not responsive to treatment with atropine.
- Sinus bradycardia is usually controlled with i.v. atropine.

Circulatory conditions and their treatment after myocardial infarction

- See Table 1 below

Table 1. Circulatory conditions and their treatment in myocardial infarction

Condition and treatment	Symptoms and signs

Normal circulation • monitoring • i.v. line (saline drop)	• heart rate and blood pressure normal • no arrhythmias • no heart insufficiency
Hyperdynamic state • beta-blocker (metoprolol, atenolol, practolol 2 - 5 mg i.v.)	• increased heart rate, high blood pressure
Neurovascular reflex (bradycardia-hypotension) • atropine 0.5 mg i.v., repeated ad 2 mg • dopamine infusion, if necessary	• usually in connection with postero-inferior infarction • bradycardia, hypotension
Hypovolaemia • 0.9% saline 200 ml in 5 - 10 min according to the response	• low blood pressure, low CVP, tachycardia • cold extremities • decreased venous distension (also jugular veins)
Severe heart failure • nitrate infusion • dopamine infusion • CPAP • treatment of pulmonary oedema	• low blood pressure • cold extremities • engorged neck veins

Treatment in hospital

Follow-up and treatment

- Pain: morphine, nitro, beta-blocker
- Blood pressure
- Skin, peripheral circulation
- Increased respiratory rate suggests cardiac insufficiency.
- Monitoring of arrhythmias
- ST segment changes
- Oxygen saturation; oxygen or CPAP
- A comfortable posture

- Informing and reassuring the patient
- Nicotine replacement therapy is started already in the hospital. Nicotine addiction may be evaluated by using the Fagerstrom test, and the planning of further treatment may be based on it.
- In an uncomplicated infarction, patients are allowed to sit as soon as they want, they can eat unassisted, and they can be helped to a portable toilet at the bedside. Intensive monitoring is usually needed for 1 - 2 days.
- The infarction is complicated and treatment lasts longer if the patient has had
    - shock
    - hypotension
    - obvious cardiac insufficiency (usually requires thrombosis prophylaxis or anticoagulation, especially if in connection with atrial fibrillation)
    - prolonged chest pain
    - serious ventricular arrhythmias
    - thromboembolic complications
    - anatomical complications (papillary muscle dysfunction or rupture)
    - pericarditis on days 2 - 4.
- Treatment of the patient in primary health care (in a primary health care hospital) is justifiable if the patient´s prognosis is otherwise poor: those who are permanent inpatients or otherwise severely disabled and for whom invasive treatment has not been planned.

Assessment of risk factors in a patient with myocardial infarction

- The most important causes of mortality are
    - reinfarction
    - cardiac insufficiency
    - arrhythmias.
- During hospitalization, a poor prognosis is indicated by
    - cardiac insufficiency and extensive infarction (EF < 25%)
    - chest pain and ischaemic ST changes (send to angiography)
    - In connection with non-Q-wave infarction, risk factors for CHD and especially diabetes mellitus.
- Evaluation of ischaemia and need for active treatment
    - risk is highest during the first few weeks and months after infarction. Therefore, at the end of the hospital treatment, an early symptom-limited exercise test is performed on many patients to estimate the need for angioplasty and coronary surgery in particular.
- For indications of coronary angiography see related EBM Guideline: **Coronary angiography and indications for CABG or angioplasty** available on the EBM Web site.

Care after myocardial infarction

Drug treatment

- ASA, beta-blocker (Level of Evidence=A; Evidence Summary available on the EBM Web site) ACE inhibitors and statins have been shown to improve the prognosis.
- Unnecessary drugs instituted during the initial phase should be discontinued already towards the end of hospital treatment, or when the patient comes to the first check-up, not on the last

day in hospital.
- Only those with cardiac insufficiency need a diuretic.
- ASA 50 - 100 (- 250) mg is given unless there are contraindications (Level of Evidence=A; Evidence Summary available on the EBM Web site).
- At least patients with hypertension, angina pectoris, ventricular arrhythmias, ischaemia during an exercise test, previous infarction, an enlarged heart, small ejection fraction or a cardiac insufficiency need a beta-blocker. In practice, these drugs are given to all patients who have no contraindications. Adequate beta-blockade is achieved when the heart rate at rest is about 60 bpm.
- Nitrate plus a beta-blocker are given to all patients with angina pectoris or ischaemia during an exercise test. Nitrate is a symptomatic drug that can often be discontinued.
- An ACE inhibitor is given to all patients with clear systolic dysfunction (ejection fraction < 40%). A milder systolic dysfunction is treated with an ACE inhibitor if the patient has cardiac insufficiency (symptomatic or asymptomatic), valvular regurgitation, hypertension, or diabetic nephropathy. The indications of ACE inhibitors have been constantly extended, and they are now given to almost every patient who has had an infarction. So-called "asymptomatic cardiac insufficiency" and even secondary prevention (according to the HOPE study) in high-risk patients have become indications [2]. ACE inhibitor therapy may be more difficult if the patient has a valvular obstruction, hypotension or uraemia. Patients on diuretics have a risk of hypotension, especially when treatment with an ACE inhibitor is started. The ACE inhibitor dose should not remain at the level of the initial dose unless hypotension and creatinine elevation prevent the reduction.
- A lipid-lowering drug (a statin) is given to all patients with serum LDL cholesterol > 3.0 mmol in spite of the diet (Level of Evidence=A; Evidence Summary available on the EBM Web site). For calculation of the level, see Program 1 of the corresponding full text guideline available on the EBM Web site.
- An anticoagulant is given if the patient has atrial fibrillation, an embolic complication or ventricular aneurysm verified by echocardiography, often also short-term in the treatment of an extensive anterior wall infarction.
- A quiet moment should be reserved for discussing life after MI and living with CAD while the patient is still in the hospital.
    - Such a discussion prevents psychological problems and disability.
    - Give instructions for possible exacerbation of the disease.
    - The motivation to quit smoking is highest:
        - nicotine replacement therapy according to individual evaluation (Fagerstrom test)
    - A cholesterol-restriction diet and/or drug treatment.
    - Exercise counselling according to individual evaluation: the patient must be able to talk while exercising.
    - Rehabilitation course
    - Secondary prevention

Sick leave

- Duration 2 - 3 months.
- Re-examination after about one month, usually within specialist health care.
    - History of symptoms: if the patient has had angina pectoris symptoms, consider testing exercise capacity, if the test has not been performed yet.
    - Remind the patient of the principles of healthy life style.
    - Serum lipids should be measured if they were high on an earlier measurement.
    - Control the adequacy of beta-blockade: target heart rate 50 - 60 bpm.

    - Possible depression should be diagnosed.
- The ability to work is evaluated before the end of the sick leave. If necessary, an exercise test is carried out to assess working ability.

Related evidence

- Glucose-insulin-potassium probably reduces mortality in acute myocardial infarction. However, its role in combination with trombolysis or acute revascularization should be determined by larger randomized trials (Level of Evidence=B; Evidence Summary available on the EBM Web site).
- There is little evidence from randomized trials of any significant further net clinical benefit from adding either subcutaneous or intravenous unfractionated heparin to the treatment of patients who are given aspirin (Level of Evidence=B; Evidence Summary available on the EBM Web site).
- Low-dose amiodarone may have a beneficial effect on total mortality after myocardial infarction, but the drug has many adverse effects (Level of Evidence=C; Evidence Summary available on the EBM Web site).
- The evidence does not support the hypothesis that verapamil use is associated with harm in patients with myocardial infarction (Level of Evidence=B; Evidence Summary available on the EBM Web site).
- Exertion-related MIs occur in habitually inactive people with multiple cardiac risk factors (Level of Evidence=B; Evidence Summary available on the EBM Web site).

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